Biopsy of the ulcerated site revealed superficial and deep chronic perivascular inflammatory infiltrate without visible amastigotes, but culture of the biopsy specimen yielded *L. braziliensis*. Sodium stibogluconate, 20 mg/(kg·d) for 10 days vs. 20 days, was administered as part of an ongoing protocol. During treatment, small satellite lesions developed around the ulcer, and two vesicular lesions arose at the base of the ulcer. By the end of treatment, the site of the lesion had improved markedly, and a follow-up examination 2 months after completing treatment demonstrated complete reepithelialization and no clinical evidence of recurrence.

Concerns regarding toxicity and reports of increasing resistance to the pentavalent antimonial compounds have led to a search for alternative treatment regimens for leishmaniasis. Lipid formulations of amphotericin B accumulate in the liver and spleen [6], which are the primary sites of infection for visceral leishmaniasis. With cutaneous disease, however, the site of infection is the dermis [7]. Panosian et al. [8] reported that liposome-intercalated amphotericin B has no effect on cutaneous leishmaniasis in immunocompetent mice and only a slight effect in the immunodeficient BALB/c strain. More recently, Yardley and Croft [9] demonstrated in a murine model that Ambisome reduced the size of lesions in experimental cutaneous leishmaniasis in a dose-dependent manner; however, all mice relapsed, suggesting that Ambisome had a suppressive rather than curative effect.

Our experience with the use of Abelcet for the treatment of cutaneous leishmaniasis in this single case concurs with the lack of curative effect seen in the murine model when lipid formulations of amphotericin B have been used to treat cutaneous leishmaniasis. On the basis of the available published data, we suggest that lipid formulations of amphotericin B are as yet insufficiently studied for the treatment of cutaneous disease and that, pending the accumulation of further data, the recommended therapy for New World cutaneous leishmaniasis continues to be one of the pentavalent antimonials. Whether higher doses, longer courses, or different lipid formulations of amphotericin B would prove to be more efficacious can only be surmised.

**References**

results of the remainder of his biochemical profile, including levels of serum amylase and red-cell transketolase, were normal. Electrocardiography showed atrial fibrillation with a rapid ventricular rate (170 beats); echocardiography showed global restrictive left ventricular dysfunction with an ejection fraction of 36%. EIA for antibodies to CMV showed a strong reaction to IgM and a weak reaction to IgG. Other serological studies for evidence of hepatitis A, B, and C; HIV infection; Epstein-Barr virus infection; Q fever; cryptococcosis; legionellosis; parainfluenza; influenza; mycoplasma; Lyme disease; mumps; chlamydiosis; leptospirosis; brucellosis; and toxoplasmosis; and the Weil-Felix reaction were all negative. Blood, sputum, and urine cultures were negative. Tests for anti-ENA and DNA antibodies were negative. Findings on ultrasonography of the abdomen were normal, apart from hepatomegaly.

Because of the severity of his clinical and echocardiographic findings, the patient began receiving IV ganciclovir (5 mg/kg q12h), and he completed a 14-day course. He became afebrile within 3 days. In addition, he was treated with digoxin, amiodarone, furosemide, and perindopril, responded well to this therapy, and converted to sinus rhythm. His symptoms abated, with the exception of some ongoing lethargy. All of his liver function indices improved, although his alkaline phosphatase level peaked at 1,076 IU/L. Pulmonary infiltrates evident on a chest radiograph resolved. Serial echocardiograms showed progressive improvement, with reversion to complete normality and an ejection fraction of 66% within 2 weeks. Therapy with digoxin, amiodarone, and furosemide was discontinued; treatment with perindopril was maintained for 6 months. Repeated serology for CMV showed an increase in the IgG reactivity and a decrease in the IgM reactivity within 2 weeks. A urine culture yielded CMV; a cytopathic effect was observed, and the infection was confirmed by immunofluorescence. He returned to work within 2 months.

The spectrum of illness caused by CMV is well documented in certain immunocompromised risk groups (e.g., transplant recipients, patients with AIDS or malignancy, and neonates). However, among immunocompetent hosts, acquisition of infection usually goes undiagnosed. A recent report and review of severe CMV infection in immunocompetent hosts described a total of 34 cases [1]. In these patients, the most commonly identified infection sites were the liver (17 cases), CNS (17 cases), and lungs (9 cases). Seven of the 34 patients were treated with either ganciclovir or foscarnet. Fifteen patients died, 14 of whom had not received either of these agents. The four cases in which there was cardiac involvement were in patients aged 14, 28, 37, and 43 years [2–5], all of whom had involvement in other organs as well. None of these patients received antiviral therapy; three died of their infections.

The patient we describe in this report was seriously ill with atrial fibrillation and a rapid ventricular rate, hypotension, and significant myocardial impairment evident on echocardiography. He also had significant involvement of his liver and lungs. Ganciclovir therapy was associated with complete resolution of his myocarditis and pneumonitis within 2 weeks and a slower resolution of his hepatitis. Perhaps his illness would have resolved without ganciclovir; however, at the time that therapy was begun, his clinical condition was deteriorating, and a marked improvement was observed within days. The major concern, which prompted the initiation of ganciclovir, was his worsening clinical and echocardiographic status. We suggest that these should be the major factors in the consideration of antiviral therapy.

This unusual case illustrates that although CMV infection in immunocompetent hosts is usually self-limiting, severe disease can occur, and specific antiviral therapy can be beneficial in such cases.

Joseph G. McCormack, Simon D. Bowler, J. Elizabeth Donnelly, and Charles Steadman
Department of Medicine, University of Queensland, and Mater Medical Centre, Mater Adult Hospital, South Brisbane, Queensland, Australia

References

To our knowledge, the phenomenon has been reported only once in the context of HIV infection [2], and in that case it occurred after the patient’s therapy had been switched to isoniazid prophylaxis. We know of no case other than the one we describe in which expansion of tuberculomas occurred concurrently to the induction of highly active antiretroviral therapy (HAART).

A 35-year-old male tested HIV positive in September 1996 (blood CD4 T cell count, 210 × 10^6/L; HIV viral load, 325,000 copies/mL). He was enrolled in a clinical trial in December in which zidovudine, 250 mg b.d., lamivudine, 150 mg b.d., and nelfinavir/placebo were administered.

Five weeks later, his HIV viral load had decreased to <400 copies/mL. However, he complained of lethargy, sweats, and weight loss. Physical examination demonstrated an 11-kg weight loss since the time of diagnosis, and there was a firm, 3 × 4 cm, nontender lymph node palpable in the right anterior cervical

Miliary Tuberculosis with Paradoxical Expansion of Intracranial Tuberculomas Complicating Human Immunodeficiency Virus Infection in a Patient Receiving Highly Active Antiretroviral Therapy

The paradoxical expansion of tuberculomas during the course of tuberculous chemotherapy has been reported occasionally [1].

Reprints or correspondence: Dr. John A. Crump, Division of Infectious Diseases, Christchurch Hospital, Private Bag 4710, Christchurch, New Zealand.

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